

# Grading and Decision-Making in (Aneurysmal) Subarachnoid Haemorrhage

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## Introduction

The interaction between medical doctors and patients in the diagnostic and therapeutic process is the result of both conscious and unconscious decision-making. When we succeed in making us aware of the decision processes, preferably based on sound facts and data, we are on the way from personal beliefs, institutional habits and educational fixations to a scientific, evidence based medical behaviour that will further improve our professional handling. Such analyses can be applied to every field in medicine, of course. In neurosurgery the field of subarachnoid haemorrhage is a great example of a battle ground where many beliefs and contradictory opinions still fight each other, resulting in so many papers on "how" and "when" to treat, to manage and to "cure" the patient. In the following survey, these problems will not be solved, but an analysis will be made of some of the general decision processes that play a part in the ultimate choice made for an individual patient. We will not go into the details of the technique of handling (clipping or coiling) specific aneurysms: these items will be dealt with separately during this course. One should be aware, however, that every step in these matters is again the result of decisions, sometimes made within fractions of seconds....

## Diagnosis

Subarachnoid haemorrhage (SAH) is a life-threatening event with an incidence of six per 100.000 people (for most countries: Finland seems to be an exception with an almost three times higher incidence rate)<sup>18</sup>. Case fatality is high, with good outcome in less than half of the patients, despite all efforts to improve the results over the last 30 years. Although it seems that only prevention of a SAH would bring about a substantial improvement in management, early recognition is still a cornerstone in the management of patients with a SAH. Therefore, the assessment of the diagnosis is the first step that should be achieved. The history of an excruciating headache of sudden onset, eventually followed by a period of unconsciousness, vomiting, and later on neck stiffness is pathognomonic for SAH. Of course, a differential diagnosis should be made, but once there is a suspicion of SAH, a straightforward diagnostic path should be followed. This consists of a CT scan of good quality, made as soon as possible. Only after a negative scan, should a lumbar puncture be performed, with a thin (22 G) needle. Timing here is crucial: in order to avoid a diagnostic dilemma, such a puncture should be done at least 12 hours after the presumed SAH. Only then can the differentiation be

made between fresh blood staining by inadvertent puncturing of a vessel, and SAH blood in the CSF. The latter will be seen as xanthochromia after centrifuging the CSF-filled tube, or even with more sophisticated analysis of blood breakdown products.

Once the diagnosis of a SAH is made, the cause has to be found: is it aneurysmal or not? Normally the patient will be transferred (or already has been) to a neurosurgical centre.

This all depends on the clinical situation of the patient. One should think ahead: if an aneurysm is found, will s/he be fit for immediate treatment? Or, will the patient anyhow be better off in a centre with daily experience in overall management of SAH? Such considerations may seem trivial, but it is the task of the

neurosurgeon to arrange for close co-operation with his/her referral centres to provide optimal management care for patients thought to have a SAH!

The gold-standard for the next step is digital subtraction angiography, DSA. Its sensitivity is high, as is its selectivity. Today there is growing experience with MR angiography, and (3D) CT angiography. Both techniques have good sensitivity and selectivity for aneurysms at least larger than 5 mm. The non-invasiveness is a strong argument, as well as the fact that, depending on the local situation, it can be more easily available, even at awkward times of day. When negative or unclear, one should proceed to DSA as yet. Even the DSA may be negative, since aneurysm rupture is only responsible for 75 – 80% of SAH, and AVM rupture for 4 – 5%. In the differential diagnosis of DSA-negative SAH the most important is perimesencephalic bleeding as a distinguishable entity<sup>1</sup>. Others include vasculitis, dural sinus thrombosis, coagulopathy, pituitary apoplexy and spinal AVM. The incidence of all these is very low, and most can be ruled out by a good quality MRI. That leaves 14 – 22% for “SAH of unknown origin”, which fortunately has a very good prognosis. The decision has to be made whether it is worthwhile in these cases to repeat the DSA at any time. Today, most centres repeat it once, after 14 – 28 days, but the gain is very low, especially when the first DSA is of

Table 1 Hunt and Hess classification of SAH

GRADES	DESCRIPTION
0	unruptured aneurysm
1	asymptomatic, or mild h.a./nuch.rigid.
1a	only fixed neuro deficit
2	moderate/severe h.a./nuch.rigid.
3	mild focal deficit, lethargy/confusion
4	stupor/moderate or severe hemiparesis, early decer.rig.
5	deep coma, decer.rig., moribund

Table 2 The grading system used by Yasargil

YASARGIL GRADE	DESCRIPTION
0 a	Unruptured aneurysm, no neurol. deficit
0 b	Unruptured aneurysm, neurol. deficit, like n III palsy, chiasma syndrome or progressive hemisyndrome (in giant an's)
I a	Asymptomatic following subarachnoid haemorrhage
I b	SAH, alert and orientated, but with focal pronounced neurological deficit like hemiparesis, paraparesis, aphasia, visual field loss (no simple lesion of cranial nerve II, III or IV)
II a	Alert, but with headache and meningism
II b	Same, with focal neurological deficit
III a	Lethargic, confused, disoriented, combative
III b	Same, with focal neurological deficit
IV	Semicomatose, response to pain, not to voice; may show extensor posturing; pupils reactive to light
V	Comatose, pupils not reactive to light, extensor posturing or no reaction to pain, failing vital signs

The rationale behind this grading system is again the relation to outcome: 0a, Ia and IIa with the best results, followed by Ib and IIb, then by IIIa, 0b, IIIb, IV and V respectively.

Table 3 The WFNS grading scale for SAH

WFNS grade	GCS score	Major focal deficit	Comment
0	-	-	intact aneurysm
1	15	absent	comp. with H&H I and II
2	13 – 14	absent	comp. with H&H III(II)
3	13 – 14	present	comp. with H&H III
4	7 – 12	present or absent	= H&H IV (III)
5	3 – 6	present or absent	= H&H V

good resolution and does not show spasm or vascular anomalies. With a negative angiogram and still strong suspicion of an aneurysm, some authors proceed with exploration – especially in the anterior communicating area – or follow further with one of the non-invasive techniques, MRA or CTA (for a recent survey<sup>19</sup>).

### Further Management

Now that a diagnosis of aneurysmal SAH has been made, the neurosurgeon will have to decide whether and how s/he should proceed with the management. There are many options, related to patient factors, physician factors, team and centre factors, as well as rules, guidelines, data and opinions in the literature and textbooks! The different aspects, important for decision-making will be dealt with in the following paragraphs.

### Grading

Assessment of the patient's condition immediately after a SAH is of the utmost importance and crucial to further management. Classification of patients should be done in a reliable way and with relevance to prognosis and outcome. In 1966, Botterell proposed a grading system with four grades. Hunt and Hess<sup>11</sup> elaborated further on this outcome-related grading, resulting in their "Hunt and Hess" grading system, which has been used world-wide for many decades. Even today, many authors still use this way of grading, although the original five-tier system was later extended to include patients harbouring unruptured aneurysms, and with the situation of a bleeding without meningeal or brain reaction, but a fixed neurological deficit only. (table 1)

Some authors preferred a more personal touch, though. Yasargil proposed a grading sys-

tem based on the revised Botterell system but recognising the importance of focal neurological deficit by adding b when existing, and a when not being apparent<sup>24</sup>. (table 2)

The problem with the grading scales has been the interobserver variability, prompted by poor definition of the terms used<sup>17</sup>. This has been proven to be worst for the Hunt and Hess scale. Terminology like "drowsiness" and "stupor" is ambiguous. Moreover, the difference between grades I and II in this scale seems to be irrelevant for outcome. Therefore, in 1988 the WFNS committee on grading proposed a system based on the Glasgow Coma Scale (GCS), a scale originally developed for trauma and proven to be very reliable as to interobserver agreement.

The major differences between WFNS and Hunt & Hess scale are given in the comments. Important is the difference between WFNS 2 and 3, based on the presence or absence of major focal neurological deficit.

Despite the scientific arguments for a preference of the use of this WFNS grading scale, adoption of this way of preoperative assessment has taken place only slowly<sup>7</sup>. (table 3)

Table 4 Glasgow Coma Scale based scale<sup>23</sup>

GCS	15	11 - 14	8 - 10	4 - 7	3
GCS based grade	I	II	III	IV	V

Table 5 Johns Hopkins GCS grading scale in SAH

GCS	15	12 - 14	9 - 11	6 - 8	3 - 5
GSC based scale	I	II	III	IV	V

Table 6 Fisher's grading system of intracranial blood on CT in SAH patients.

Fisher grade	blood on CT (< 5 days after SAH)
1	no subarachnoid blood detected
2	diffuse or vertical layers < 1 mm thick
3	localised clot and/or vertical layer → 1 mm
4	intracerebral or intraventricular clot with diffuse or no SAH

Interestingly, in a recent elaborate study, Takagi and co-workers<sup>23</sup> analysed their data on a very large series of aneurysm patients using the GCS in a very detailed breakdown of the figures. They made a reasonable argument for a GCS-based grading with breakpoints between GCS 3 and 4; 7 and 8; 10 and 11; and 14 and 15 respectively. This leads again to a five-tier scale (table 4), but different from the WFNS scale. In doing so, the outcomes related to the different scales became significantly discernible from each other, which was not the case when the WFNS scale was applied. (table 4)

A similar refining of the WFNS grading scale was published by a group from Johns Hopkins two years earlier<sup>20</sup>, also based upon a compression of the GCS into five grades, with a greater predictive value regarding outcome than the H&H or WFNS scales (table 5).

The differences with the Tagaki scale are the breakpoints, as can easily be seen. Comments on both GCS-based grading systems concern for example the apparent difference between H&H grade I, II and III patients, all of whom may enter grade I in these newer grading scales. Another problem arises with a confused patient who might enter H&H grade III, but is now in grade II (GCS 14), similarly to a patient who does not open his eyes spontaneously but furthermore intact (also GCS 14), again apparently quite different! The coming years will teach us whether such a further fine tuning

compared to the WFNS grading scale has relevance to prognosis of outcome and hence in the decision-making in aneurysm treatment.

In assessing patients with a proven aneurysmal SAH, not only the clinical grading is important, but also the amount of subarachnoid (and/or intracerebral) blood. A reliable and universally used system for this is the grading system developed by Fisher et al<sup>6</sup> (1980, soon after the introduction of CT scanning!) (table 6).

The positive correlation between the amount of intracisternal blood on the first CT scan after SAH and the subsequent development of vasospasm and delayed ischemic deficit gives this grading scale a prominent role in decisions on individual management. This correlation is strongest for Fisher grade 3. Transcranial Doppler measurements have, in a general, confirmed such a correlation, even when vasospasm did not become symptomatic (most recent study and survey:<sup>14</sup>).

Results of management have to be measured in a reliable way as well. Although some authors have used personalised 4- or 5- point scales, the Glasgow Outcome Scale (GOS) has been adopted widely over the last 20 years. This five-point scale was developed for the follow-up of head-injured patients, but proved to be applicable in stroke and SAH patients as well<sup>15,22</sup>. Rather similar is the so-called modified Rankin scale. The scale is practical and reproducible, but one should be aware that the even-

#### The Glasgow Outcome Scale

Grade	Neurological status
1	Good recovery; patient can lead a full and independent life with or without minimal neurological deficit
2	Moderately disabled; patient has neurological or intellectual impairment but is independent
3	Severely disabled patient, conscious but totally dependent on others to get through daily activities
4	Vegetative survival
5	Dead

tual individual outcome is influenced by many other factors. Subtle concentration disturbances, problems with memory and certain changes in character can make an enormous difference to a patient's life when compared to his situation before the SAH, even when s/he is scored in GOS 1! Definite neuropsychological damage varies between 11% and 60%, depending on how detailed patients are tested<sup>12</sup>. A differentiation should be made between effects of the SAH itself, the (peri-)operative management, and the reflection of premorbid weakness (related to the "event", not specifically to the SAH)<sup>10</sup>.

### Selection and Timing

The next important and intermingled steps in the management are the decisions on:

- can/should we treat the aneurysm in this patient? (different from: "taking care of him/her"!).

- when should we treat the aneurysm?
- how should we treat it?
- can I (we) treat it?

The goal of treating the aneurysm is primarily to prevent rebleeding, the most life-threatening complication after a SAH with a mortality chance of around 80%. Maximal frequency of rebleeding is on the first day (4%), then 1.5% daily for 13 days, resulting in 15 – 20% rebleeding within 14 days, and 50% within six months. Thereafter the risk tapers off to 3% per year. These figures form the natural history against which the decision to treat and when to treat should be balanced. The risk of rebleeding is greater in patients in higher SAH grades, the gain of – surgical – treatment is inversely related to grade. Before 1980, surgery was postponed in most centres to a "quiet period", at least 14 days after SAH. As a result, many patients died while waiting. With the growing experience in microsurgical techniques and aneurysm surgery, more and more centres dare to operate "early" – within three days – with still improving results. Today, at least in the larger centres, the attitude is to operate early on patients in grade I – III, and to postpone surgery in the others. The more difficult cases (posterior circulation, giant aneurysms) are postponed as well. In poor grade patients with beginning hydrocephalus, ventricular drainage may bring the patient one grade up, making him fit for surgery. Some centres favour surgery

on even poor grade patients, saving some of them without enlarging the poor outcome (vegetative state) category. Other centres still continue to postpone surgery in every SAH patient. So there are still controversies on these issues, as there are many arguments in favour and against early surgery! It is obvious that a lot more can be said about the decision process in this stage: co-morbidity, age, resulting life expectancy, experience of the surgeon(s)' own track records! – and the wishes of the patient and/or relatives, they all play a role.

In recent years, the alternative to surgical repair – endovascular treatment – has come into the armamentarium, changing the whole field of this discussion (see next). It should be recognised that there is no Class I data to support one of the decision options to date (for a concise survey see Greenberg<sup>8</sup>).

### Clip or Coil

The boom in endovascular treatment – presumably with Guglielmi coils – has changed the scope in timing and decision. Details will be presented elsewhere in this course. For decision-making it is important to know results so far, and to realize that a clip – provided that it is properly placed – occludes definitively, while for coiling there is not that same proof. On the other hand, coiling results are very promising, improving over recent years, with acceptable complication rates and apparently no negative effect on the frequency of hydrocephalus or vasospasm. Studies are underway to show long-term results and outcome. Small neck aneurysms can be occluded by coiling in over 70%, treatment-related complications occur in less than 10%, with morbidity under 2%. Experience varies per centre; a state of the art for indications has not been established yet. In most centres today the coiling of posterior aneurysms – until now very often postponed for the first 12 days after SAH – has become the upfront treatment within 72 hours after the bleed. Its role for other indications will become established in the near future<sup>2,5</sup>.

### Post-Treatment Care

Once the aneurysm has been taken care of, the patient will encounter many serious problems and threats: delayed ischaemic deficits (DID) from vasospasm, hyponatremia, hydro-

cephalus, and more. The use of nimodipine has been adopted world-wide. Other treatment and care modalities (HHH, for example) are largely based on the belief and not on proof of its effectiveness. Day to day monitoring is the key issue of course. Despite many drug trials, no specific medication has proven to be significantly effective in preventing DID except nimodipine, which is not always enough, though. Decisions have to be made on guidelines and experience rather than on evident data (see the major textbooks). There is place for new prospective studies in this field!

### Multiple Aneurysms

Many reports can be found on the concomitant treatment of more than one aneurysm. A CT scan does not always help to decide which one has bled; this may be a problem when the aneurysms cannot be reached all by one approach. Coiling might be an option here as well. Once again, one should be aware of the natural history, which for the non-bleeding aneurysms is different from the one that has bled (see next paragraph), before planning a second operation to reach all aneurysms.

### Unruptured Aneurysms and Screening

All neurosurgeons involved in aneurysm surgery are aware of the often contradictory data concerning the risk of rupture in unruptured aneurysms, either symptomatic or incidentally found<sup>13,16</sup>. The ISUIA study has given a lot of data, showing much lower rupture risks than thought before, and higher surgical complication rates than neurosurgeons want to think of<sup>21</sup>!

Risks far below 1%, especially for aneurysms under 10 mm, have already led to new "feelings" among neurologists and neurosurgeons about whom to treat and whom not. At this stage, it is too early to make definite statements on risks, the results of the continuing ISUIA study have to be awaited, and one should not take part in self-fulfilling prophecies by completely changing the indications and decision routine already. It is evident that low rupture risks for non-bleeding aneurysms have enormous consequences for the usefulness of screening in populations, and even among relatives of SAH patients. This will be dealt with elsewhere.

### Decision-Making: Decision Analysis as a Science

To end this contribution, it should be emphasized that in daily practice decisions, although possibly and preferably based on the best evidence available, harbour a certain aspect of "feelings and experiences". This will certainly result in a good (or the best!) treatment option for the individual patient, but the way along which such decisions are reached is often difficult to follow by others. By the use of decision analysis methods, such decision-making can be formalized, leading to comprehensible decision trees<sup>9</sup>. Components of such trees are dichotomies and trichotomies, for example surgery / no surgery, followed by complications / no complications, ending in results after the different major branches of the tree: well, disabled, dead.

By filling in known probability figures, like natural history aspects (annual risk of rupture; mortality and morbidity of rupture) and treatment aspects (surgical morbidity, mortality), patient's co-morbidity, age and life expectancy, and correcting for quality aspects (including the fear of the patient who knows that he has an aneurysm!) one can "fold back" the figures and reach so-called Quality Adjusted Life Years.

Although models always oversimplify reality, an assumption on the "gain" in QUALY for either of one option can be made, for example surgery or no surgery in a patient harbouring an incidental aneurysm, or screening or no screening in families with a history of SAH, or in the general population<sup>3,4</sup>.

### Conclusion

For a scientific basis to the best treatment of patients with saccular aneurysms, and especially those with aneurysmal SAH, a sound and reliable grading system should be applied, as well as logical management steps based on data of results and final outcome for the various treatment options.

Unfortunately, not all facts and figures available have that level of evidence. Recognition of the process of decision-making, enlightening the weak and strong parts of it, may help to improve decision-making itself, and thereby enhance the overall management of patients with saccular aneurysms.

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### Further reading

Most neurosurgical textbooks dedicate large chapters to almost all aspects of aneurysms and SAH. Specific books that are very helpful in grasping the “scope” and the decision aspects in SAH are:

Bryce Weir: *Aneurysms affecting the nervous system*. 1987, Williams & Wilkins, Baltimore. ISBN 0-683-08925-0  
 J.Bederson (ed): *Subarachnoid haemorrhage: pathophysiology and management*. 1997, AANS Neurosurgical Topics Series. ISBN 1-879284-43-X  
 I.A.Langmoen et Al: (eds) *Neurosurgical management of aneurysmal subarachnoid haemorrhage*. 1999, Acta Neurochirurgica Suppl 72, Springer Verlag Wien.

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